SPLENECTOMY FOR BANTI'S DISEASE,

FOLLOWED BY THROMBOSIS OF THE RIGHT INTERNAL JUGULAR, SUBCLAVIAN, AND INNOMINATE VEINS. RECOVERY.

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THAT splenic anæmia has been but imperfectly understood may be readily seen by a reference to the designations of this symptom complex given by various authors. Eichorst described it as splenic pseudoleukæmia, Cohnheim as pseudoleukæmia, Banti and Gretsel as splenic anæmia, Gaucher as idiopathic hypertrophy of the spleen, De Bove as hypertrophy of the spleen without leukæmia. That this still remains an obscure question is apparent, for Osler,[1] in his latest publication, states as follows: "The question of the existence of a separate malady—Anæmia Splenica—is still in what might be called the inquisitive or the tentative stage."

Sippy[2] as well as Osler,[3] in a series of cases of splenic anæmia, describe a condition more or less characteristic, having the following clinical picture: The disease occurs in young adult life, developing insidiously, oftentimes without any manifestation, until the sudden appearance of hæmorrhage, which generally proceeds from the stomach. This symptom may be the first trouble experienced by the patient and causes him to seek medical advice. The disease is frequently accompanied by dizziness and a slight pallor, due to anæmia. In a number of Osler's cases the anæmia was not profound.

The attention of the patient is often attracted to a tumor in the left side, which may be the first indication of the disease. Nausea, vomiting, epistaxis, profuse and protracted diarrhœa frequently occur. As the tumor increases in size, the patient complains of a dragging sensation in the side, but few expe-

rience any pain. Bronzing of the skin is frequent (melanoderma). This was present in about 60 per cent. of Osler's cases. Harris [4] reports the disappearance of this pigmentation subsequent to a splenectomy for splenic anæmia. There is no lymphatic involvement which differentiates it from Hodgkin's disease. The liver may or may not be enlarged. Œdema and ascites may develop. Hæmorrhages are very frequent, death at times resulting from profuse bleedings or exhaustion.

The blood findings are fairly constant. Reds about 3,500,000; a low color index showing a diminution of hæmoglobin disproportionate to the number of red cells (secondary anæmia of the chlorotic type). In the greater number of cases leucopænia is present, generally below 5000. There are no nucleated red cells, nor does the differential count reveal anything of significance: no myelocytes, excluding leukæmia. In several of Osler's cases ascites was present.

Banti [5] describes a condition which develops in three stages, and is characterized by cirrhosis of the liver, splenomegaly, and ascites. In the first stage, anæmia and splenic enlargement are present, the anæmia being secondary to the enlargement of the spleen. The duration of this period varies between three and ten years, and is followed by a second stage marked by a diminution in the quantity of urine, and an increase in bile pigment and urates: this continues for several months, then follows the third and most characteristic stage, distinguished by the development of ascites, which appears insidiously and is unaccompanied by pain. The anæmia increases, and at times there is an evening rise of temperature. Banti states that there is a slight increase of white corpuscles, but most observers agree that leucopænia usually exists. Death occurs within a year after the disease has fully developed (Senator [6]).

To Banti belongs the credit of having simplified our knowledge of the pathology of these conditions. It was he who first claimed that the cirrhosis of the liver was secondary to the enlargement of the spleen, which is contrary to what obtains in primary cirrhosis of the liver. He also maintained

that the splenic enlargement was due to a toxæmia, the products of which, passing through the liver, cause an hyperplasia of the connective tissue, such as occurs in the liver of alcoholics. As the disease is accompanied by ascites, the toxæmic theory would have material support, for the spontaneous disappearance of the ascites in Banti's disease is evidence that portal obstruction, in consequence of interstitial hepatitis, does not exist.

Barr [7] advances the following theory in reference to Banti's disease; that it is due to a vasomotor paresis of the splanchnic area, which arises from a disease of the visceral sympathetic ganglia. This results in an engorgement of the abdominal viscera, especially the liver and spleen, with hæmolysis as a consequence, and which is followed by oligochromia and oligocythæmia. The increased blood supply leads to fibrosis and lessened function; it also causes the peritoneal effusion, which is in no way dependent upon portal obstruction. The vasomotor paresis which lowers blood pressure leads to a retention of blood in the portal circulation; hæmorrhage and impaired digestion being the result of this engorgement.

Infection has been considered an etiological factor, but bacteriological investigations up to this time have resulted negatively. In the etiology, the digestive tract must be considered; to this Senator calls attention on account of the diarrhœa.

The development of the disease, with its consequent ascites, at once attracts attention to the liver, which we ordinarily consider the causative factor in the development of ascites. Pousset,[8] in an analytical study of the relative size of the liver and spleen, states that in cirrhosis of the liver the spleen is moderately enlarged compared to the size of the liver, while in Banti's disease and splenic anæmia the spleen may often exceed the liver in size.

Senator [9] reports a case of Banti's disease accompanied by marked ascites, which necessitated the removal at different intervals of twenty-five litres of fluid. Eventually the fluid did not reaccumulate, entirely disappearing. He remarks that if interstitial hepatitis were present, it would be unusual for the

fluid to disappear in this manner, as this is not the history of ascites complicating cirrhosis of the liver.

Senator accounts for the disappearance of the fluid by reasoning that enlarged mesenteric glands which have been reported to exist, may open a collateral lymphatic circulation, and so relieve this stasis of lymph. Hæmorrhagic tendency, the most important symptom, remains to be mentioned. Bleeding may occur in the form of epistaxis, a very common manifestation, or it may appear as hæmatemesis. Purpura, also hæmorrhage into the vitreous body, are occasionally seen. The gravest of all, however, is the bleeding that proceeds from the stomach, and which may result in death. In all probability, some of the cases of so-called cirrhosis of the liver, with enlargement of the spleen, complicated by hæmatemesis, have in reality been either splenic anæmia or Banti's disease. If this assumption is correct, these conditions will be oftener recognized as their true nature becomes better known. The bleeding may be the first symptom of the disease, and the anæmia its consequence.

Senator [10] has collected all of the cases (up to the publication of his paper) where it was possible to obtain a complete report; many of these have been considered splenic anæmia, but which he thinks should be classified as Banti's disease. He states that the blood findings are fairly uniform, presenting the combination of oligocythæmia, oligochromia, and leucopænia. This combination, however, is found in well-developed cases only, for the leucocyte count is more or less dependent upon other factors, such as hæmorrhage, infection, etc. Here a leucocytosis due to a pneumonia or other infectious process would cause alterations in the blood. In one of Harris's cases there was a leucocytosis of 28,000.[11]

A recapitulation of the blood findings shows

First; oligocythæmia. There is a diminution of the red cells, the average being about 3,500,000.

Second; oligochromia. The low percentage of hæmoglobin is fairly constant, and is marked by a low color index; in pernicious anæmia the contrary obtains.

Third; leucopænia. This is quite constant. Osler reports that out of thirteen cases, nine gave a leucocyte count of less than 5000; in six of these cases the leucopænia was extreme. The red cells and the differential count reveal nothing of diagnostic value. Senator states, however, that the cases on record have been too few to warrant the statement that the blood findings in Banti's disease are quite characteristic.

In conclusion, it is evident that Senator considers Banti's disease as a disease characterized by an enlargement of the spleen without lymphatic involvement, tendency to hæmorrhage, oligocythæmia, oligochromia, leucopænia, and accompanied by ascites. Cirrhosis of the liver may or may not be present. He considers the disease as splenic anæmia with ascites.

Osler's [12] deductions upon this symptom complex are apparent from the following definition of anæmia splenica chronica which he offers: "A chronic affection, probably an intoxication of unknown origin, characterized by a progressive enlargement of the spleen, which cannot be correlated with any known cause, as malaria, leukæmia, syphilis, cirrhosis of the liver, etc. (primary splenomegaly); anæmia of a secondary or chlorotic type (leucopænia); a marked tendency to hæmorrhage, particularly from the stomach; and in many cases a terminal stage with cirrhosis of the liver, jaundice, and ascites (Banti's disease)."

That surgery of the spleen has made great strides during the past decade may be readily seen by a reference to the monograph of Bessel Hagen [13] upon this subject, and which still remains the most important contribution to the literature. His statistics include a number of surgical diseases of the spleen, which, however, have no place here; but in the disease that concern us the following figures demonstrate how materially the death-rate has fallen during the past decade.

In fifteen cases of extirpation of a wandering spleen due to malaria there was one death (6.6 per cent.). In sixty-nine cases of extirpation of the spleen for malarial hypertrophy there were six deaths (8.7 per cent.). The mortality was

in reality 23.4 per cent.; but by eliminating cases in which there had been errors in technique and diagnosis, together with improper selection of cases for operation, Hagen figures the mortality in favorable cases to the above. In twenty-eight cases of wandering spleen with idiopathic hypertrophy there were two deaths (7.1 per cent.). The statistics in general show that out of 131 splenectomies for different causes there were sixteen deaths (12.2 per cent.). As stated before, errors which might have been obviated, such as slipping of ligature, sepsis, etc., have been eliminated. With these not eliminated, the mortality remains 18.9 per cent. against 42.2 per cent., the mortality before 1890.

The number of cases of splenic anæmia and Banti's disease which have been treated by splenectomy prove how much can be accomplished by surgical measures, for, if not treated in this manner, the disease usually terminates fatally. It is just here that great stress should be laid concerning an accurate diagnosis; for in the experience of many, patients have died of recurrent hæmorrhages, where the usual diagnosis of cirrhosis of the liver has been made upon the clinical findings, namely, cirrhosis of the liver, enlarged spleen, and hæmatemesis being present. In some of these cases life might have been saved by a correct understanding of the pathology, together with an accurate diagnosis, followed by early surgical interference. In subjecting these cases to operation, the diagnosis is of the greatest importance, for the mortality in splenectomy has been materially increased as a result of improper selection of cases. Mariagliano [14] and Terrille [15] report a series of sixteen cases of Banti's disease treated by splenectomy with three deaths. In this series there was an uncertainty of diagnosis in two cases, death in both instances resulting from uncontrollable bleeding. In the other case death was due to a perforation of the uterus during curettage for an abortion, the consequence of a splenectomy.

Warren [16] reports a splenectomy performed for splenic anæmia, and Harris [17] reports two cases operated for the same condition. All recovered. Immediately after splenectomy

for these conditions, there is an increase of the red and white corpuscles without a corresponding increase in the hæmoglobin; following this there is a diminution of the reds which persists for several months, when the reds gradually reach a high figure. The high leucocyte count as well as the low color index persist for a long time. Harris calls attention to the large percentage of eosinophiles (14.4 per cent.) that was observed in one of his cases; in this case a number of red cells (hazy) and many microcytes were found as late as twenty months after the operation.

The following case of Banti's disease presents several points of interest.

E. S., aged twenty-seven years; male; weight in health, 185 pounds; habits regular; does not drink to excess. Family history negative. No history of malaria or syphilis. In 1894 patient developed an acute appendicitis with perforation, for which he was operated by the writer. Two days subsequent to the appendectomy, a toxæmia developed in consequence of an accumulation of fluid in an acutely dilated stomach. The patient vomited great quantities of an offensive blackish fluid, and appeared to be "in extremis;" temperature, 104.5° F.; pulse, 150. As a last resort, the stomach tube was introduced, when great quantities of this fluid were evacuated. The temperature and pulse dropped immediately. Convalescence uninterrupted. This case had several points of interest which were at the time reported.

Up to 1896 the patient had remained in good health, playing cricket, etc. One day, without warning, he was suddenly seized with a hæmorrhage from the stomach, vomiting large quantities of red blood, also passing blood by rectum. During this time he had never experienced any pain in the epigastrium, nor had he any cough. Anæmia was the sequel of this bleeding; the patient, however, recovering completely. In 1897, one year later, he had a similar experience, supposed at the time to be due to a strain. This attack was more severe than the previous one, and confined the patient to bed for six weeks; but he subsequently regained his usual health. In May, 1901, being four years later, the patient again vomited blood profusely for three days; recovery again complete. On November 28, 1901, he was in perfect health,

stating that he never felt better in his life, when he was suddenly seized with a hæmorrhage from the stomach of a most alarming nature. He was seen by the writer thirty-six hours later and found to be almost exsanguinated. This bleeding recurred at intervals for three days, the blood being bright red and the stools tar-colored. No pain.

November 30, 1901. Face very anæmic, lips blanched, pupils moderately dilated, reacting to light. Pulse, 96; full but compressible. Hæmic murmur. Abdomen: dulness continuous with the splenic dulness, extending from the upper seventh rib sixteen centimetres downward in the midaxillary line, and continuous with the heart dulness. It also reaches to the same height posteriorly. The mass is easily palpated and slightly movable; notch can be felt. Liver, upper sixth, costal margin. Urine, 1080 cubic centimetres in twenty-four hours. Urea, 2¾ per cent.; traces of albumen, quantities of granular casts, some few red blood-corpuscles.

Blood examination: hæmoglobin, 30 per cent.; leucocytes, 5000; reds, 2,500,000.

December 2. Vomited matter contains no blood.

December 3. Dulness remains the same. Vomited 420 cubic centimetres of blood. Hæmoglobin, 25 per cent.; reds, 2,800,000; whites, 28,000; temperature, 101.8° F. Slate-colored stools. (Note the leucocytosis and accompanying temperature together with the slate-colored stools, all of which might indicate an infection emanating from the intestinal tract.) Patient very weak; 200 cubic centimetres of a 2½ per cent. gelatin in normal salt solution were injected, after which the hæmorrhage did not recur.

December 4. Patient improving.

December 12. Dulness extends from the upper seventh seventeen centimetres downward in the midaxillary line, and across from the costal angle to the median line.

January 15. Hæmoglobin, 25 per cent.; reds, 2,800,000; whites, 4000. Patient suffers greatly from insomnia due to an intense pain between the scapulæ, necessitating the use of very large doses of morphine without material relief (up to four grains hypodermatically within four hours). This pain caused the patient more suffering than all of the other symptoms combined. It persisted about one month and then disappeared.

Liver-dulness: upper sixth, costal margin (no enlargement),

splenic dulness upper seventh rib, seventeen centimetres in the midaxillary line. Dulness extends to eleven centimetres on either side of the umbilicus and moves upon change of position (ascites). No anasarca. Patient has been passing slate-colored stools for three weeks: they are not clay-colored, but are bluish in appearance.

January 25. Fluid in the abdomen continues to increase. The veins lateral to the umbilicus are visibly distended; no distention of the veins surrounding the navel. Paracentesis abdominis, three litres.

February 9. Fluid reaccumulating; paracentesis about to be repeated, when the disappearance of the distended abdominal veins was observed, and, as this pointed to the re-establishment of the portal circulation, it was not carried out. The subsequent history confirmed this belief, for the fluid gradually disappeared, and did not reform.

Blood: reds, 2,537,000; whites, 4200; hæmoglobin, 22 per cent.; plasmodium none.

Differential count: polymorphonuclear neutrophiles, 86.8 per cent.; small lymphocytes, 6.06 per cent.; large lymphocytes, 6.31 per cent.; eosinophiles, .75 per cent.

Five hundred leucocytes counted; one megaloblast was seen (large nucleated red cell). There is a marked diminution of hæmoglobin in each cell. A very decided variation in the size of the red cell exists, many microcytes being present; also a few megalocytes and poikilocytes.

March 16. Reds, 2,074,284; whites, 1300 (note the low white count); hæmoglobin, 44 per cent.; (Fleischl), 38 per cent.; (chloroform benzol), plasmodium none.

Differential count of leucocytes: polymorphonuclear neutrophiles, 79 per cent.; large lymphocytes, 10.5.; small lymphocytes, 4.5 per cent.; eosinophiles, 6 per cent.

No myelocytes were seen. The small lymphocytes invariably show perinuclear basic granules; this, however, has no special significance. The red cells all stain feebly, and are variable in size; numbers of microcytes, megalocytes, and poikilocytes being present. Very few normal red cells are found. One large nucleated red cell (megaloblast) was observed.

March 23. Reds, 3,066,000; whites, 1450; hæmoglobin, 45 per cent.; (Fleischl), 45 per cent.; (chloroform benzol); plas-

modium none. Examination of stained specimens showed the same condition as existed one week before, with the exception that there is less variation in the size of the red cells.

March 26. Patient rapidly improving, has gained twenty pounds. Liver, upper sixth, costal margin, nine centimetres in the mammillary line; spleen extending almost to the iliac crest, seventeen centimetres in the midaxillary line; painless; notch easily felt.

March 29. Reds, 3,387,480; whites, 1925; hæmoglobin, 43 per cent.; plasmodium none.

Differential count of leucocytes: polymorphonuclear neutrophiles, 77.5 per cent.; large lymphocytes, 13.4 per cent.; small lymphocytes, 6.7 per cent.; eosinophiles, 1 per cent.

A good many megalocytes (3 to 4 per cent.) as well as microcytes (25 to 30 per cent.) are present, together with a moderate number of poikilocytes. There are a few shadow cells. The red cells more nearly approach the normal than at any previous examination. One large nucleated red cell was seen.

April 6. Reds, 3,156,666; whites, 1550; hæmoglobin, 40 per cent. (chloroform benzol).

Differential count: polymorphonuclear neutrophiles, 88.5 per cent.; small lymphocytes, 6.5 per cent.; large lymphocytes, 2 per cent.; eosinophiles, 3 per cent.

The red cells stain better than on previous occasions, and the number of normal cells is larger than heretofore, the number of shadow cells and microcytes being less.

That the foregoing case is one of Banti's disease the writer believed to be evident, each symptom conforming to the picture described by Banti and other observers.

After a careful consideration of the indications for the removal of the spleen as a life-saving expedient, and in view of the history of the case, together with the blood findings, splenectomy was advised and acceded to. Patient in excellent condition, notwithstanding his 40 per cent. of hæmoglobin; deeply bronzed; was accosted on the street by friends, who inquired of him if he were not jaundiced. The writer remarked that the patient resembled a Chinaman. This melanoderma, which was apparent all over the body, seemed to be growing darker. Liver and spleen as before noted. Urine: no albumen or sugar; casts absent; urea, 2.8 per cent.

April 8, 1902. Under chloroform narcosis an incision was made in the left semilunar line, extending from the left costal angle to the level of the navel. The blood which flowed from the incision was seen to be very light in color and quite watery. The spleen was readily exposed and palpated in its entirety. Diaphragmatic adhesions were absent, but the splenodiaphragmatic ligament was readily felt connecting the organ to the diaphragm. Great difficulty was experienced in delivering the spleen on account of the shortness of the pedicle, which consisted of the peritoneal fold, including the splenic vessels, constituting the gastrosplenic ligament. The first attempt at ligation was followed by an alarming hæmorrhage due to the tearing of one of the large splenic sinuses; this hæmorrhage could only be controlled after the spleen had been delivered, which became possible when the ligament extending to the diaphragm was tied off. This permitted the delivery of the upper end of the organ, after which the entire spleen was lifted out and turned from left to right, the pedicle being exposed from below. Warren, in his splenectomy for splenic anæmia, had a similar experience. It was necessary, however, to enlarge the incision, by making a supplementary incision extending from the middle of the vertical incision horizontally to the left for a distance of about three inches. Warren extended his incision to the right, cutting across the rectus. The pedicle was ligated in sections tied double and cut between; extra ligatures applied to the individual vessels, to avert secondary hæmorrhage; chromic gut was used. The vessels of the greater omentum were enormously enlarged, but were not interfered with on account of the necessity of maintaining a collateral circulation, possibly obviating a recurrence of ascites. There was considerable hæmorrhage from oozing low down from the diaphragm, near the spinal column. This was controlled by gauze packing, which was allowed to remain thirty-six hours. No shock from the operation, notwithstanding the fact that there was considerable loss of blood. Color good when removed from the table. Duration of operation, one and one-half hours, most of the time lost in controlling oozing. Pulse 72, good. Compress shows considerable oozing not sanguinolent. 4 P.M. Pulse, 82; temperature, 100.6° F.; urine, 390 cubic centimetres by catheter.

April 10, 6 A.M. Pulse, 92; temperature, 99.6° F.; respiration, 22; urine, 1140 cubic centimetres in twenty-four hours.

Some of drain removed. At this time the melanoderma seemed to be fading, more especially on the body; this was remarked by every one who saw the patient. Ears and lips a brighter red than before the operation. 6 P.M. Pulse, 100; temperature, 100.6° F.; respiration, 24. Whites, 24,780; reds, 3,100,000; hæmoglobin, 50 per cent.

April 11, 6 A.M. Pulse, 94; temperature, 100° F.; respiration, 24; urine, 1740 cubic centimetres in twenty-four hours. Drain removed. Bowels moved with calomel.

Blood examination: reds, 3,100,000; whites, 15,500; hæmoglobin, 55 per cent.

The red cells in general stain better than before the operation; there are a few pale cells as well as a few of abnormal size and shape. The general impression received being that the blood is much nearer normal than at any previous examination.

Differential count of leucocytes: polymorphonuclear neutrophiles, 81 per cent.; small lymphocytes, 3 per cent.; large lymphocytes, 16 per cent.; no eosinophiles; 500 white cells counted. A considerable number of anomalous forms are seen, and which can be separated into two classes, viz.:

First, polymorphonuclear cells without or with few granules (quite unusual); second, large lymphocytes with deeply indented nucleus, apparently about to divide (transitional form).

April 12, 6 A.M. Temperature, 100.6° F.; urine, 960 cubic centimetres; hæmoglobin, 55 per cent. 6 P.M. Temperature, 101° F.

April 13, 6 A.M. Temperature, 100° F.; urine, 900 cubic centimetres in twenty-four hours. 6 P.M. Temperature, 101.2° F.

April 14, 6 A.M. Temperature, 100.6° F.; urine, 900 cubic centimetres in twenty-four hours; urea, 3 per cent. 6 P.M. Temperature, 102.2° F.; pulse, 96.

Blood examination: Reds, 3,112,000; whites, 12,000; hæmoglobin, 57 per cent. (von Fleischl).

Differential count of leucocytes: polymorphonuclear neutrophiles, 73.9 per cent.; large lymphocytes, 18.1 per cent.; small lymphocytes, 0.7 per cent.; eosinophiles, 1.9 per cent.

Twelve nucleated cells counted: normoblasts, 10; megaloblast, 1; microblast, 1.

April 15, 6 P.M. Temperature, 101° F.; urine, 720 cubic

centimetres in twenty-four hours; urea, 3 per cent. Patient convalescent, wants to know when he can get up. There is no evidence of enlargement of either lymphatics or of the thyroid gland.

Reds, 3,064,000; whites, 8000; hæmoglobin, 55 per cent.

No differential count was made. The various forms of leucocytes, however, seemed to be in the same proportion as at the last examination. Special staining failed to show the presence of any mast-cells. The red cells were mostly fairly normal in size and staining properties. A few normoblasts were seen, no distinct megaloblasts.

April 21. Hæmoglobin, 55 per cent.

Leucocytes: polymorphonuclear, 86 per cent.; large lymphocytes, 11.25 per cent.; small lymphocytes, 1.6 per cent.; eosinophiles, 1 per cent.

Red cells: a few macrocytes and microcytes are seen, also a very few poikilocytes. In counting 700 leucocytes, sixteen nucleated reds are seen, classified as follows: twelve normoblasts, two microblasts, two megaloblasts. The latter were rather doubtful, and were classed as megaloblasts on account of the staining properties of the nucleus rather than on the size of the cell. The large majority of reds are normal or nearly so, as seen in the stained specimen.

April 25. Reds, 2,922,000; whites, 12,400; hæmoglobin, 56 per cent.

Leucocytes: polymorphonuclear, 79 per cent.; mononuclear large, 10.5 per cent.; mononuclear small, 9.5 per cent. Of the latter, an unusually large number are rather medium sized and difficult to classify as large or small. In counting 500 whites, one mononuclear and one polynuclear basophile were seen.

Red cells: only two nucleated cells were seen, one being a normoblast and one a microblast. There is a decided reduction as compared to the last examination. Macrocytes and microcytes are not numerous; the large majority of red cells being normal in size and staining reaction.

April 26, 6 A.M. Temperature, 99° F.; P.M., 100° F. Patient complains of pain on the right side of the neck. Sternomastoid very sensitive to pressure, postcervical glands enlarged. Pain extends from the right shoulder to the mastoid region. Axillary glands not palpable; cubital gland on the right side much

enlarged; sublingual glands swollen and painful. Thyroid gland enlarged and painful.

April 30. Neck very painful; inguinal glands indurated and enlarged. A large indurated mass is felt under the sternomastoid on the right side; left side unaffected. Temperature, 101.8° F.; 6 P.M., 102.2° F. Swelling extends from the sternoclavicular articulation to the ear. No superficial induration.

May 1. Temperature, A.M., 102.2° F.; pulse, 100. Neck, shoulder, and arm enormously swollen. The diagnosis of a deep-seated cervical abscess was made and operation decided upon. The patient's condition was quite alarming; sensorium dulled.

Blood examination prior to the operation was as follows: Hæmoglobin, 50 per cent.; red cells not estimated; leucocytes, 30,500.

Differential count: polymorphoneutrophiles, 85 per cent.; large lymphocytes and transition forms, 12 per cent.; small lymphocytes, 2 per cent.; eosinophiles, 0.5 per cent.; mast-cells, 0.5 per cent.; myelocytes none. Nine hundred white cells were counted. Nucleated red cells were seen as follows: five normoblasts and one microblast. The red cells in general seem to be not far from normal, very few of abnormal size and shape are seen. Under ether narcosis, an incision was made along the inner border of the sternomastoid, but no pus was found. The internal jugular was easily exposed, and seen to be firmly distended with a clot. On account of the mottled appearance of the wall of the vessel, it was incised and the clot laid bare. No evidence of pus could be discovered. The vein was then followed from the mastoid down to the sternal articulation, a thrombosis of the entire vessel being disclosed. An incision, such as is usually done in ligating the subclavian, was made above the clavicle, in the hope that the entire thrombosed vessel might be excised; but the exposure of the subclavian vein as well as the right innominate vein behind the sternoclavicular articulation showed how futile that procedure would be. Both the subclavian and the innominate veins, which were distended with clot, could be easily followed behind the sternoclavicular articulation up to the superior vena cava as easily as it is followed upon the injected cadaver. Further manipulation was desisted from; the internal jugular was tied and cut at the point of incision and the wound closed. The patient reacted well from the operation, and after two days

the swelling began to subside, and within two weeks it had almost disappeared. Coincident with the subsidence of the swelling, the superficial veins of the shoulder and back on the right side became much dilated, evidently due to the establishing of a collateral circulation; this dilatation was present after one year, but has now about disappeared.

That we had a thrombosis of the right innominate vein was evident at the time of operation; that the left innominate was not involved was apparent by the absence of all manifestations on the left side; hence we are warranted in assuming that the superior vena cava remained patent. From this time on convalescence was unimpeded, excepting for the persistence of severe sweating, which gradually ceased.

May 7. Reds, 2,800,000; whites, 32,000; hæmoglobin, 50 per cent.

Differential count: polymorphonuclear, 80.5 per cent.; small lymphocytes, 2 per cent.; large lymphocytes, 13.5 per cent.; mast-cells, 1 per cent.; eosinophiles, 3 per cent.

Nucleated red cells were fairly numerous in counting 400 leucocytes, and consisted of thirty-five normoblasts and two microblasts. Some of the normoblasts showed dividing trilobate and quadrilobate nuclei.

May 20. Reds, 3,700,000; whites, 9300; hæmoglobin, 61 per cent.

Differential count of whites: polymorphonuclear neutrophiles, 61.5 per cent.; large lymphocytes and transition forms, 22.5 per cent.; small lymphocytes, 12.5 per cent.; eosinophiles, 3.5 per cent.; mast-cells, 0.5 per cent.

One nucleated red cell was seen, that a normoblast. Considerable variation in the size of the red cell exists.

July 6. Red cells, 4,268,000; whites, 7600; hæmoglobin, 63 per cent.

Differential count: polynuclear neutrophiles, 67.5 per cent.; large lymphocytes and transition forms, 23 per cent.; small lymphocytes, 8 per cent.; eosinophiles, 1 per cent.; basophiles, 0.5 per cent.

Red cells: nucleated forms, a few are present; four or five to one cover slip preparation, all being normoblasts. The only deviation from the normal being a considerable variation in the size of the cells, and failure of some to take the stain well (dimi-

nution of hæmoglobin). After this the blood became quite normal and has remained so up to date. The patient has gained about sixty pounds, has had no further hæmorrhages, and says that he feels stronger and better than he has felt for years. There is no glandular enlargement nor is there any swelling of the right arm. Melanoderma entirely disappeared.

The following blood examination was made June 3, 1903, this being about fourteen months subsequent to the splenectomy. Red cells, 6,400,000; whites, 9500; hæmoglobin, 96 per cent.

Differential count (500 leucocytes); polymorphoneutrophiles, 81.8 per cent.; large mononuclear and transitional forms, one per cent.; small mononuclear, 15.4 per cent.; eosinophiles, 8 per cent.

Pathological report of excised spleen made by Dr. Arthur L. Fisher, pathologist to the Mount Zion Hospital.

The spleen has been preserved in 4 per cent. formaldehyde solution since the day of the operation. It now measures 21.5 centimetres by 14.5 centimetres by 8 centimetres, and its weight, wet, 1350.0 grammes.

Paraffin sections were made and stained in hæmatoxylin and eosin, eosin and methylene blue, and in carbol fuchsin and hæmatoxylin (for tubercle bacilli). The sections stained for the tubercle bacilli did not show any, and those stained in eosin and methylene blue failed to reveal the presence of any other micro-organism. None of the sections stained well.

The capsule of the spleen is very much thickened, as is the connective tissue throughout the spleen. Around the larger vessels this is particularly noticeable; some of the largest vessels are surrounded by a dense band of thick fibrous tissue. In a few of the medium-sized vessels just below the capsule the blood still remains; in other places it has escaped,—probably simply run out when the spleen was cut, as there is practically nothing to indicate that the blood supply has been interfered with to any extent.

There is a great deal of iron free blood pigment (hæmatoidin) throughout the spleen, both in the central and in the peripheral portions, but more in the periphery than in the centre. The pigment is deposited mainly in the interstices between the cells. There is very little evidence of active phagocytosis; only here and there are seen a few polymorphonuclear leucocytes containing pigment and other detritus. There are none of the large

endothelial phagocytic cells to be seen. The Malpighian bodies are apparently not increased in size nor relatively in number. Absolutely there is probably a considerable increase in number. The vast majority of these bodies are normal in appearance; a few, however, are undergoing hyaline degeneration at their centres, and occasionally one is seen in which almost the entire body has undergone hyaline degeneration. The increase in the size of the spleen is to be accounted for mainly by an increase in the number of certain of its component cells, notably the elongated more or less fusiform fibrous tissue cells, the endothelial cells, and to a lesser extent the lymphoid cells. The cells are closely crowded, apparently obliterating the smaller spaces and pressing the walls of the sinuses together. In the central portion it appears as though the spaces are completely lined with endothelial cells. This, in connection with the increase of the supporting fibrous tissue, gives the spleen a glandular appearance. The capillaries in the fibrous tissue are very abundant.

The endothelial cells are rather small for cells of this kind. The nucleus is slightly more solid than is usually seen in endothelial cells. It is somewhat larger than the nucleus of a lymphocyte, and is surrounded by a cell body of a rather poorly staining protoplasm.

The larger vessels show quite a marked thickening of the intima. The main changes are increase in the fibrous tissue; proliferation of endothelial cells; moderate amount of hyaline degeneration of the Malpighian bodies.

SUMMARY OF INTERESTING FEATURES IN THE FOREGOING CASE.

First, the long period of personal observation.

Second, the invasion of the disease, preceded by an infectious appendicitis.

Third, the intervals of perfect health, interrupted by profuse bleedings.

Fourth, the blood findings, which corresponded in every particular to Senator's description.

Fifth, the exacerbation of temperature and the accompanying leucocytosis, both indicative of some form of infection.

Sixth, the slate-colored stools and the diarrhœa, evidence of a digestive disturbance.

Seventh, the severe pains between the shoulder-blades, and the enormous quantities of morphine given necessary to relieve pain.

Eighth, the disappearance of the distended abdominal and chest veins, followed by the spontaneous disappearance of the ascites.

Ninth, the splenectomy followed by the rapid increase in the percentage of hæmoglobin.

Tenth, the appearance of the normoblasts and the megaloblasts in the blood subsequent to the splenectomy.

Eleventh, the rapid regeneration of a very abnormal blood.

Twelfth, the disappearance of the melanoderma following the operation.

Thirteenth, the thrombosis of the right innominate, subclavian and internal jugular veins, and the recovery of the patient from the same.

In conclusion, it might be said that Banti's disease forms a distinct chapter in the surgery of the spleen, and that it offers a favorable prognosis if operated early, death resulting if it is allowed to go unoperated, the gravity of the operation increasing "pari passu" with the size of the spleen.

I take this opportunity of expressing my sincerest appreciation to Dr. G. W. McCoy, of the United States Marine Hospital Service, for the splendid service he has rendered in the accurate observation of the blood in the above case.

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